Original Article

MEDICINE PALLIATIVE CARE

DOI: 10.47582/jompac.1217035

J Med Palliat Care 2023; 4(1): 21-27

Association of serum uric acid/albumin ratio with completely occluded infarct-related artery in patients with non-ST-segment elevation myocardial infarction

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Cite this article as: Karayiğit O. Association of serum uric acid/albumin ratio with completely occluded infarct-related artery in patients with non-ST-segment elevation myocardial infarction. J Med Palliat Care 2023; 4(1): 21-27.

ABSTRACT

Aim: Infarct-related artery (IRA) patency before primary percutaneous coronary intervention (pPCI) is linked to improved clinical outcomes and lower mortality in patients with acute coronary syndrome. The purpose of this research was to examine the association between serum uric acid/albumin ratio (UAR) and IRA patency in patients with non-ST-segment elevation myocardial infarction (NSTEMI).

Material and Method: We evaluated 430 consecutive patients with NSTEMI in total retrospectively. The study population was divided into 2 groups according to the IRA patency as assessed by the degree of Thrombolysis in Myocardial Infarction (TIMI) flow before pPCI. As a result, completely occluded IRA was defined as TIMI grade 0-1, while patent IRA was defined as TIMI grade 2-3.

Results: IRA was found to be occluded in 110 (25.5%) patients prior to the procedure. UAR level (p=<0.001) was found to be higher among the patients with IRA occlusion when compared to the patent group. Regression analysis revealed that UAR (OR:3.125; 95% CI:1.186-8.232, p<0.001), left ventricular ejection fraction (OR:0.917, 95% CI:0.885-0.951, p<0.001) and culprit artery diameter (OR:0.917, 95% CI:0.885-0.951, p<0.001) were independent predictors for an occluded IRA. An UAR cut-off value of >1.40 was detected to prognosticate the occluded IRA with 62.7% sensitivity and 63.8% specificity (AUC: 0.722, 95% CI: 0.671-0.773, p<0.001).

Conclusion: UAR is an independent predictor of preprocedural IRA patency in patients with NSTEMI. Thus, UAR may be an easily accessible parameter to diagnose high-risk NSTEMI patients who would benefit from an immediate invasive strategy (<2 hours).

Keywords: Serum uric acid/albumin ratio, infarct-related artery, non-ST elevation myocardial infarction

INTRODUCTION

ST-segment elevation myocardial infarction (STEMI) is characterized by fully enclosed or almost fully enclosed occlusion of the infarct-related artery (IRA). Early restoration of coronary flow in IRA is associated with better clinical outcomes and lower mortality (1). Therefore, primary percutaneous coronary intervention (pPCI) is currently preferred as the best treatment process for STEMI (2). As a matter of fact, non-ST-segment elevation myocardial infarction (NSTEMI) represents a wide range of clinical conditions that are usually associated with atherosclerotic plaque rupture and result in intermittent or incomplete thrombotic occlusion of IRA (3). In particular, it has been hypothesized that the adverse clinical outcomes associated with STEMI are due to complete occlusion of IRA (4). Moreover, previous studies have reported that approximately 30% of NSTEMI patients had completely occluded infarct-related arteries, as they experienced angiographic features similar to those of STEMI patients (5,6). Finally, each patient with NSTEMI underwent a postponed revascularization procedure based on the absence of ST elevation on the electrocardiogram (ECG), despite a completely occluded infarct-related artery (6).

As the most common and important protein of human serum, serum albumin (SA) plays a vital role in many important biological functions. Albumin is a circulating antioxidant protein and its decreased synthesis and increased catabolism indicate an increased inflammatory state (7). The pathophysiological mechanism of hypoalbuminemia in coronary artery disease (CAD) can be essentially related to its inefficacy in performing antioxidant, anti-inflammatory and anti-platelet aggregation activities, which consequently results in intensified blood viscosity, decreased endothelial function, oxidative stress, and narrowing in coronary artery due to large numbers of platelets (8). On the other hand, as a byproduct of purine metabolism, serum uric acid (UA) promotes the probability of atherosclerosis,

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Received: 09.12.2022 **Accepted:** 07.01.2023

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as high levels contribute inevitably to the occurrence and prognosis of coronary artery disease (9). Moreover, UA has been shown to be a mediator of inflammation, endothelial dysfunction, and vascular disease (10). Latest researches have revealed that serum uric acid/albumin ratio (UAR) as a novel inflammatory marker is associated with severe clinical symptoms in patients with acute coronary syndrome (ACS) (11,12).

Although the presence of a fully occluded IRA is relatively common in NSTEMI, limited data are available on factors related to IRA patency. Therefore, we tried to evaluate the association between serum UAR and IRA patency in patients with NSTEMI before pPCI.

MATERIAL AND METHOD

The study protocol was approved by the Yozgat Bozok University Clinical Researches Ethics Committee (Date: 13.10.2022, Decision No: 2017-KAEK-189_2022.10.13_01). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki. This current study was a retrospective analysis; therefore, patients were not required to give informed consent.

Study Population

The cross-sectional and single-centered study encompassed 430 consecutive patients in total that were admitted to the department of emergency with the diagnosis of NSTEMI and underwent pPCI from May 2021 to September 2022. NSTEMI was diagnosed as the absence of ≥ 2 mm ST segment elevation consistent with myocardial infarction in the adjacent chest leads and the absence of ≥ 1 mm ST segment elevation in the two standard leads with new left bundle branch block and positive markers of cardiac necrosis (3). Exclusion criteria include the inability to reach serum UA or albumin levels, history of coronary artery bypass grafting, STEMI, hypo/hyperthyroidism, acute decompensated heart failure, severe hepatic or renal dysfunction, hematological or autoimmune diseases, malignancies, presence of active infection, chronic inflammatory diseases, use of medication which may increase serum UA levels or patients with high serum UA such as gout, patients with unidentified IRA or multiple IRAs, and myocardial obstruction within non-obstructive coronary arteries.

Each patient involved in the research was evaluated in terms of age, gender, and cardiovascular risk variables based on their folders recorded in the hospital database.

Laboratory Measurements

Blood sampling of peripheral venous catheters was provided from the patients by means of atraumatic puncture from the antecubital vein during diagnosis, prior to sending them to the catheter lab examination. We used Beckman Coulter AU 5800 autoanalyzer in order to measure ratios of blood biochemical elements such as creatinine, potassium, uric acid, lipid panel, albumin, and sodium. Complete blood count variables were analyzed using an automated blood cell counter (Beckman Coulter LH 750; Beckman Coulter Inc., USA), while UAR was measured through the division of the serum UA level by the albumin level. The neutrophil/lymphocyte ratio (NLR) was calculated by dividing the total neutrophil count by the lymphocyte count using the same blood samples collected at admission. In addition, platelet/lymphocyte ratio (PLR) was measured through the division of the platelet count by the lymphocyte count.

Transthoracic echocardiography (Vivid 7 GE Medical System) was applied on each patient just followed by pPCI in the coronary intensive care unit, whereas left ventricular ejection fraction (LVEF) was identified through Simpson's method.

Angiographic Analysis

We used the Standard Judkins technique (Expo; Boston Scientific Corporation, Natick, Massachusetts, USA) and Siemens Axiom Sensis XP device (Munich, Germany) so as to observe the coronary angiography. Each coronary artery was visualized in at least two perpendicular planes. Just before the intervention, all patients received 180 mg of ticagrelor or 600 mg of a loading dose of clopidogrel and 300 mg of acetylsalicylic acid. During the PCI procedure, a bolus of 70 IU/kg unfractionated heparin was administered to the patients. The use of the stent type (naked or drug-coated) and the glycoprotein IIb/IIIa receptor inhibitor tirofiban was left to the operator's choice. PCI processes were conveyed using iopromide (low osmolarity and nonionic contrast agent) in accordance with the NSTEMI guidelines declared by the European Society of Cardiology (3). All patients enrolled in the study were required to undergo cardiac catheterization within 72 hours of admission together with PCI, surgical revascularization, or medical management, depending on the assessment of the operator in charge.

Just after completing patient recruitment, two experienced interventional cardiologists who were blinded to the study recorded all coronary angiographic images digitally in order to assess the rate of IRA flow. The culprit artery was defined mainly by angiographic findings [presence of obvious or suspected thrombus, ulcerated or ruptured plaque, and Thrombolysis in Myocardial Infarction (TIMI) flow grade ≤ 2]. In addition, ECG and echocardiogram recordings supported angiography data in the assessment of IRA in case of uncertainty.

Prior to coronary intervention, the antegrade flow of IRA for each patient was visually calculated according to

the TIMI study classification (13). Thus, TIMI flow was rated as follows: TIMI flow Grade 0: no antegrade flow; TIMI flow Grade 1: partial contrast penetration beyond an occlusion with incomplete distal filling; TIMI flow Grade 2: patent epicardial artery with opacification of the entire distal artery (but with delayed contrast filling and/or washout); TIMI flow Grade 3: patent epicardial artery with normal flow (13). Based on the findings of this rating system, the research groups were separated into two different groups those with TIMI 0-1 flow with a completely occluded IRA (n=110) and those with TIMI 2-3 flow with a patent IRA (n=320). There was no difference among interventional cardiologists for calculated TIMI flow grades.

Statistical Analysis

All findings were obtained through the use of the SPSS 22.0 Statistical Package Program for Windows (SPSS Inc., Chicago, IL, USA), followed by testing the data for normality using the Kolmogorov-Smirnov test. Normally distributed continuous data were shown as mean±standard deviation, while non-normally distributed continuous data were displayed as median (interquartile range 25-75). Qualitative variables were displayed in numbers and percentages. We compared parametric continuous variables using the Student t-test, whereas non-parametric continuous variables were evaluated through the Mann-Whitney U test. In addition to comparing categorical variables using chi-square and Fisher's exact test, we used the receiver operating characteristic (ROC) analysis to define the optimum cutoff level of UAR to predict occluded IRA. As multivariate logistic regression was used to determine the independent predictors of occluded IRA, a two-sided $p < 0.05$ was considered statistically significant for all analyzes.

RESULTS

The research encompassed 430 patients (male: 301; mean age: 62.8±10 years) in all, as the study groups were divided into two different groups according to the baseline antegrade flow of the IRA including 110 patients (male: 80; mean age: 61.3±10.7 years) with totally occluded IRA (pre-PCI TIMI flow 0-1) and 320 patients (male: 221; mean age: 63.3±9.8 years) with patent IRA (pre-PCI TIMI flow 2-3). Fundamental attributes and laboratory parameters of the study population are listed in **Table 1**. There was no statistically remarkable difference among patients with and without occluded IRA in terms of age, gender, hypertension, the frequencies of diabetes mellitus, dyslipidemia, familial history, tobacco use, and history of CAD (**Table 1**). Additionally, SBP and LVEF were significantly lower while length of hospital stay and peak troponin I level were remarkably higher in obstructed IRA group (**Table 1**). Moreover, NLR [3.7 (2.05-5.96) vs. 2.71 (1.9-4.26); p=0.005] and UAR [1.53±0.26 vs. 1.29±0.29; p<0.001] were remarkably higher in the occluded IRA group when compared to the patent IRA group (**Table 1**).

Angiographic characteristics of the study population regarding the presence or absence of occluded IRA are listed in **Table 2**. Here, no significant difference was detected in terms of IRA lesion length and number of diseased vessels between the groups (**Table 2**). However, time from admission to PCI was significantly higher while culprit artery diameter was remarkably lower in patients with occluded IRA (**Table 2**). On the other hand, the left circumflex artery (LCx) was the major culprit artery (43.6%) in the occluded IRA group, while the left anterior descending artery (LAD) was more common (49.1%) in the patent IRA group. Additionally, proximal localization for culprit lesion was more common in the patent IRA group than that of the occluded IRA group (51.9% vs. 40%; p=0.036).

Agents that were found to be noteworthy in univariate analyses were assessed by means of the multivariate logistic regression model so as to determine independent predictors of occluded IRA. As a result, it was observed that the UAR was an independent predictor of IRA patency. Moreover, we determined that values of LVEF and culprit artery diameter served as independent predictors of occluded IRA (**Table 3**).

As revealed by the ROC curve analysis; the cut-off value of 1.40 for UAR predicted the occluded IRA with a sensitivity of 62.7% and specificity of 63.8% (AUC: 0.722; 95% confidence interval: 0.671-0.773; p<0.001) (**Figure**).

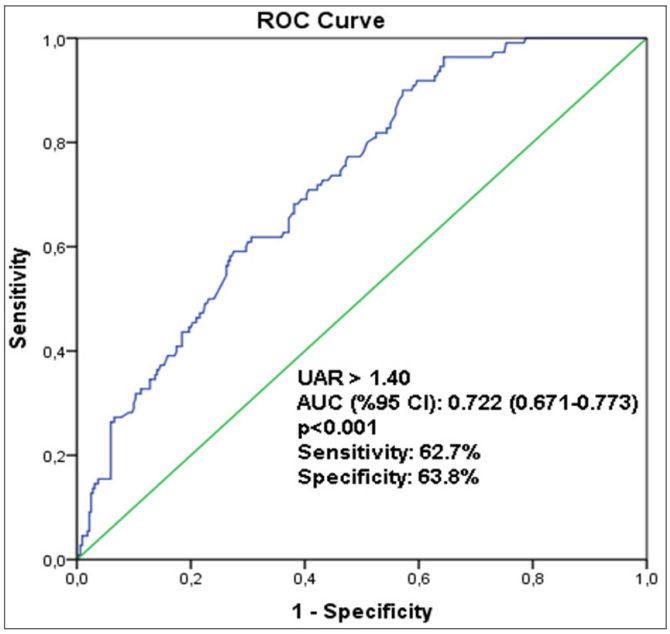

Figure. The receiver operating characteristic (ROC) curve analysis of serum uric acid/albumin ratio for the prediction of occluded infarct-related artery

Table 1. Baseline characteristics and laboratory parameters of the study groups

Total study population (n=430)			
Variables	**Occluded IRA group (n= 110) Pre-PCI TIMI flow 0/1**	**Patent IRA group (n=320) Pre-PCI TIMI flow 2/3**	**p value**
Baseline characteristics			
Age, years	61.3±10.7	63.3±9.8	0.099
Male gender, n (%)	80 (72.7)	221 (69.1)	0.547
Diabetes Mellitus, n (%)	41 (37.3)	121 (37.8)	0.920
Hypertension, n (%)	53 (48.2)	173 (54.1)	0.320
Dyslipidemia, n (%)	45 (40.9)	142 (44.4)	0.578
Current smokers, n (%)	59 (53.6)	140 (43.8)	0.077
Previous CAD, n (%)	24 (21.8)	81 (25.3)	0.521
Family history of CAD, n (%)	20 (18.2)	62 (19.4)	0.888
Left ventricle EF, %	48.5±8.2	53.5±6.5	**<0.001**
SBP at admission, mmHg	125±21	129±19	**0.010**
Heart rate at admission, bpm	76±15	77±14	0.900
Length of hospital stay, day	2.55±1.01	2.36±1.00	**0.038**
Laboratory parameters			
Glucose, mg/dl	135 (112-195)	125 (104-183)	0.080
Creatinine, mg/dl	0.85±0.23	0.83±0.22	0.822
Sodium, mmol/L	136 (135-139)	136 (134-138)	0.839
Potassium, mmol/L	4.3±0.99	4.24±0.83	0.338
Uric acid, mh/dl	6.13±1.04	5.33±1.23	**<0.001**
Albumin, g/dl	4.06 (3.8-4.2)	4.1 (3.9-4.3)	**0.002**
Total cholesterol, mg/dl	193 (164-230)	190 (163-221)	0.443
HDL-C, mg/dl	40.8±9.5	42.2±12.7	0.313
LDL-C, mg/dl	125 (106-159)	124 (101-152.5)	0.317
Triglycerides, mg/dl	137.3±92.9	145.4±124.4	0.805
WBC count, x103/μL	9.5 (8-12)	9.05 (7.4-10.9)	**0.018**
Neutrophil count, x103/μL	6.5 (5.2-8.8)	5.6 (4.6-7.6)	**0.003**
Lymphocyte count, x103/μL	1.9 (1.3-2.5)	2.1 (1.5-2.8)	0.140
Hemoglobin, g/Dl	14.4±1.6	14±1.6	0.051
RDW, fL	13.8 (13.4-14.5)	13.9 (13.4-14.5)	0.693
Platelet count, x103/μL	232 (197-259)	234 (196-276)	0.600
Peak troponin I, ng/L	10927 (4723-19816)	2313 (637-7479)	**<0.001**
NLR	3.7 (2.05-5.96)	2.71 (1.9-4.26)	**0.005**
PLR	120.5 (85.2-178.4)	109.7 (80.6-158.3)	0.174
UAR	1.53±0.26	1.29±0.29	**<0.001**

All values are expressed as mean±standard deviation, median (25th and 75th interquartile range), and number (%). Abbreviations: CAD: Coronary artery disease; EF: Ejection fraction; HDL: High-density lipoprotein; IRA: Infarct-related artery; LDL: Low-density lipoprotein; NLR: Neutrophil to lymphocyte ratio; PLR: Platelet to lymphocyte ratio; RDW: Red cell distribution width; SBP: Systolic blood pressure; TIMI: Thrombolysis in Myocardial Infarction; UAR: Uric acid to albumin ratio; WBC: White blood cell. p values in bold signify statistically significant differences.

Table 2. Angiographic features of all patients according to the presence or absence of occluded IRA

	Total study population (n=430)		
Variables	**Occluded IRA group (n= 110) Pre-PCI TIMI flow 0/1**	**Patent IRA group (n=320) Pre-PCI TIMI flow 2/3**	**p value**
Infarct-related artery, n (%)			
LAD	36 (32.7)	157 (49.1)	**0.004**
LCX	48 (43.6)	92 (28.7)	**0.005**
RCA	26 (23.6)	71 (22.2)	0.792
Proximal location for culprit lesion, n (%)	44 (40)	166 (51.9)	**0.036**
Extent of CAD, n (%)			
Single-vessel disease, n (%)	47 (42.7)	135 (42.2)	0.921
Two-vessel disease, n (%)	43 (39.1)	130 (40.6)	0.822
Three-vessel disease, n (%)	22 (20)	57 (17.8)	0.669
Culprit artery diameter, mm	2.70±0.43	2.96±0.50	**<0.001**
IRA lesion length, mm	29.7±13.7	27.7±12.3	0.198
Time from admission to PCI, hour	3.20±2.44	3.67±2.41	**0.021**

All values are expressed as mean±standard deviation and number (%). Abbreviations: CAD: Coronary artery disease; IRA: Infarct-related artery; LAD: Left anterior descending; LCX: Left circumflex; PCI: Percutaneous coronary intervention; RCA: Right coronary artery; TIMI: Thrombolysis in Myocardial Infarction. p values in bold signify statistically significant differences.

Table 3. Univariate and multivariate logistic regression analysis for assessment of predictors of occluded infarct-related artery

Variables	Univariate analysis		Multivariate analysis	
	OR (95% CI)	p value	OR (95% CI)	p value
Systolic blood pressure	0.987 (0.976-0.999)	0.028	0.993 (0.980-1.005)	0.258
NLR	1.072 (1.019-1.128)	0.008	1.046 (0.942-1.160)	0.400
PLR	1.002 (1.000-1.005)	0.038	0.999 (0.994-1.004)	0.647
UAR	4.238 (1.516-12.872)	<0.001	3.125 (1.186-8.232)	<0.001
LVEF	0.914 (0.886-0.942)	<0.001	0.917 (0.885-0.951)	<0.001
Culprit artery diameter	0.260 (0.143-0.473)	<0.001	0.307 (0.164-0.574)	<0.001
Proximal location for culprit lesion	0.618 (0.398-0.960)	0.032	0.605 (0.355-1.031)	0.065

Abbreviations: CI: Confidence interval; LVEF: Left ventricular ejection fraction; NLR: Neutrophil to lymphocyte ratio; OR: Odds ratio; PLR: Platelet to lymphocyte ratio; UAR: Uric acid to albumin ratio.

DISCUSSION

Through this research, we evaluated the connection between UAR and patency of IRA in patients with NSTEMI, revealing that UAR is a remarkable and independent marker of preprocedural IRA patency in individuals with NSTEMI.

The number of NSTEMI patients is increasing worldwide, and approximately 30% of them are thought to experience complete obstruction of a coronary artery (5,6). In our study, the prevalence of occluded IRA was detected as 25.5%, consistent with the literature. These patients experience more severe clinical symptoms and display a poor prognosis than those with non-occlusive culprit arteries. Meta-analyses have suggested that delayed invasive approach can have adverse cardiovascular consequences on prognosis (5,6). As suggested by Stone et al. (1) found, patients exhibiting IRA TIMI flow grade 3 can expect improved results in terms of heart failure, preservation of EF, and clinical endpoint at 6 months when compared to those with TIMI grades 0 to 2. Advantages of early IRA patency include a reduction in enzymatic infarct size, fatal arrhythmic events, and in-hospital mortality (14).

The emergence of an occluded IRA in patients with NSTEMI may not be merely detected through clinical or electrocardiographic measurements. It has not been figured out yet why the typical ST segment elevation does not manifest itself despite complete occlusion of the artery in patients with NSTEMI. Decreased sensitivity of the standard 12-lead ECG in detecting acute occlusion changes in the inferolateral distribution, the presence of good collaterals, acute total occlusion in an area with double blood supply, and chronic total occlusion, which are misclassified as acute, may be listed as possible mechanisms (5,6).

Previous studies in the literature have shown differences in the anatomical distribution of the culprit artery between the occluded IRA and patent vessels in the NSTEMI groups. Khan et al. (6), evaluating the data obtained from 6 studies, reported that RCA was the most common culprit artery. In contrast, Huang et al. (5) reported that LCx was the most commonly involved artery in NSTEMI patients with occluded IRA. In addition, in another study led by Hwang et al. (15), it was shown that the distal vascular bed is more frequently involved as being the anatomical location of the lesions in patients with occluded IRA. Similarly, our study has revealed that proximal vessel involvement is more common in patients with patent IRA.

Albumin level is an independent and readily available cardiovascular prognostic biomarker. A reduction in SA has been found to be associated with poor in-hospital survival, in-stent restenosis, and coronary artery disease severity (16,17). Furthermore, decreased SA concentrations are closely linked to the emergence and growth of coronary atherosclerosis (18). Loss of the antioxidant properties of albumin can lead to an advanced risk of clotting in the coronary capillary lumen (19).

Serum UA is a byproduct of purine metabolism (19), thus it can initiate local inflammatory responses by forming monosodium urate crystals in various tissues. It has been reported that these activities involving UA crystals are significantly increased in patients with CAD (9). Human atherosclerotic plaque contains significant amounts of UA, thus high serum UA can promote thrombus formation via purine metabolism (20). In addition, UA can give rise to oxidative stress and induce inflammation, vasoconstriction, and endothelial dysfunction (21). As observed in patients with hyperuricemia, almost all elements mentioned above can play a significant role in the progression of atherosclerosis and can potentially result in the progression of CAD.

As the role of UAR in patients with cardiovascular disease has been studied, it can clearly indicate the presence of inflammation and oxidative stress. Kalkan et al. (12) found that STEMI patients with a higher UAR have an increased risk of death. Çakmak et al. (22) showed that a high UAR level is a more reliable predictor of the extent of CAD (using the SYNTAX score) in patients with NSTEMI than the C-reactive protein/albumin ratio. In another recent research, a high UAR level was reported to be an independent marker of the emergence of contrast-induced nephropathy after pPCI in patients with STEMI (23).

Taking into account the relevant findings, it can be suggested that UAR has a close relationship with the coronary flow in patients with NSTEMI. Thereby, we showed that UAR was remarkably higher in NSTEMI patients with an occluded IRA, and a high UAR level was an independent predictor of an occluded IRA. Yet, further studies can clarify whether a higher UAR value in NSTEMI patients is an indicator of IRA.

Nevertheless, this study has some restrictions. Firstly, the single-center, retrospective design may have led to biases. Therefore, it failed to fully control for confounding factors, including undocumented drug history, nutritional status linked by serum albumin level, and comorbidities. The small sample size is a second and important limitation. Thus, the values regarding the incidents and the data obtained did not allow us to evaluate measures reflecting NSTEMI prognoses, such as TIMI score, GRACE score, Killip class, or BNP levels. Also, we were unable to collect data on ECG changes at admission, so it was unclear whether posterior ECG leads were applied or not. We calculated UA and SA levels merely at admission, thus the use of a spot laboratory value prevented us from using values obtained over some time. Another limitation is that the findings are not predictive for patients with other acute coronary syndromes because only NSTEMI patients were included.

CONCLUSION

We revealed that there is a remarkable association between UAR and pre-procedural infarct-related arterial patency in NSTEMI patients. UAR was an independent predictor of an occluded IRA in patients undergoing PCI for NSTEMI. Thus, UAR may be an easily accessible parameter to diagnose high-risk NSTEMI patients who would benefit from an immediate invasive strategy (<2 hours).

ETHICAL DECLARATIONS

Ethics Committee Approval: The study protocol was approved by the Yozgat Bozok University Clinical Researches Ethics Committee (Date: 13.10.2022, Decision No: 2017-KAEK-189_2022.10.13_01).

Informed Consent: Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: No conflict of interest was declared by the author.

Financial Disclosure: The author declared that this study had received no financial support.

Author Contributions: The author declares that he has responsible for the design, execution, and analysis of the paper and that he has approved the final version.

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